Amendments to the Specification:

On page 7, replace the paragraph beginning at line 7 and ending at line 14 with the following:

In another aspect, the invention includes a method of identifying a compound effective to enhance hypoxic or ischemic damage in a cell. A ψδRACK ~~agonsit~~ agonist peptide is contacted with a δPKC peptide containing a RACK binding site in the presence and absence of a test compound. The test compound is identified as being effective to enhance ischemic damage if (i) binding in the presence of the test compound is decreased relative to binding in the absence of the test compound, or (ii) the catalytic activity of the δPKC in the presence of the test compound is increased relative to the catalytic activity in the absence of the test compound.

On page 14, replace the paragraph beginning at line 15 and ending at line 31 with the following:

In studies performed in support of the present invention to identify peptide sequences for activation and inhibition of δPKC, the sequence of δPKC was compared to the sequence of θPKC, since of the three other novel PKC isozymes, δPKC is most similar to θPKC with a 52% identity of amino acid sequence (Osada, S.-I *et al.*; Baier, G. *et al.*). It was also assumed that each PKC isozyme should interact with a different RACK. Since the first variable (V1) domain of δPKC contains the RACK-binding site (Johnson *et al.* 1996a) regions least similar to θPKC may be involved in RACK binding. Fig. 1 compares the sequences of the V1 domain of rat δPKC (SEQ ID NO:2; accession no. KIRTCD) and mouse θPKC V1 domain (SEQ ID NO:3, accession no. Q02111). Three regions in the V1 domain of δPKC were identified with only ~10% identity to θPKC. These regions are indicated in Fig. 1 by the bars above the sequence of δPKC and are referred to herein as δV1-1 having a sequence identified herein as SEQ ID NO:4 (SFNSYELGSL), δV1-2 having a sequence identified herein as SEQ ID NO:5

(ALTTDRGKLV), and ψδRACK having a sequence identified herein as SEQ ID NO:6 (MRAAEDPM). Not shown in Fig. 1 is yet another sequence identified from the δPKC sequence for testing of its activation or inhibition of δPKC. This sequences is identified as SEQ ID ~~NO7~~ NO:7 and is referred to herein as δV1-5.

Replace the paragraph beginning on page 14 at line 32 and ending on page 15, line 15 with the following:

As described in Example 1, the δV1-1 and ψδRACK peptides were analyzed to determine whether the peptides had activity, and if so, whether the activity was as an agonist or an antagonist of δPKC. As will be shown, δV1-1, δV1-2 and δV1-5 are δPKC antagonists and ψδRACK is a δPKC agonist. In these studies, the δV1-1 and ψδRACK peptides were modified with a carrier peptide by cross-linking via an N-terminal Cys-Cys bond to the *Drosophila* Antennapedia homeodomain (SEQ ID NO:8; Théodore, L., *et al.*; Johnson, J. A. *et al.*, 1996b). In other studies, not described here, the peptide was modified with Tat (SEQ ID NO:9) or with polyarginine (Mitchell *et al.*, 2000; ~~Rolhbard~~ Rothbard *et al.,* 2000) and gave results similar to those described herein. Details of the study are set forth in Example 1. In brief, the Antennapedia-conjugated peptides were introduced to cardiac cells at a concentration of 500 nM in the presence and absence of phorbol 12-myristate 13-acetate (PMA) or in the presence of each other. Translocation of δPKC isozyme was assessed by Western blot analysis cystosolic and particulate fractions of treated cells. Subcellular localization of δPKC isozyme was assessed by immunofluorescence by probing the blot with anti-δPKC, anti-αPKC, and anti-ε PKC antibodies. Translocation was expressed as the amount of isozyme in the particulate fraction over the amount of isozyme in non-treated cells. The results are shown in Figs. 2-4.

Replace the paragraph on page 15, lines 16-23 with the following:

Figs. 2A-2B show the results for the cells treated with δV1-1 in the presence (+) and absence (-) of PMA. Fig. 2A is the Western blot autoradiogram of soluble (S) and

particulate (P) cell fractions after treatment with the peptide and after probing with anti-δPKC and anti-εPKC antibodies. Fig. 2B shows the translocation of δPKC expressed as the amount of isozyme in the particulate fraction over the amount of isozyme in non-treated cells. The δV1-1 peptide inhibited PMA-induced δPKC translocation. In other studies ~~no~~ not shown here, the δV1-1 peptide did not inhibit the translocation of εPKC or αPKC.

Replace the paragraph on page 22, lines 10-25 with the following:

For δV1-1, potential modifications include the following changes shown in lower case: tFNSYELGSL (SEQ ID NO:34), aFNSYELGSL (SEQ ID NO:35), SFNSYELGtL (SEQ ID NO:36), including any combination of these three substitutions, such as tFNSYELGtL (SEQ ID NO: 37). Other potential modifications include SyNSYELGSL (SEQ ID NO:38), SFNSfELGSL (SEQ ID NO:39), SNSYdLGSL (SEQ ID NO:40), SFNSYELpSL (SEQ ID NO:41). Other potential modifications include changes of one or two L to I or V, such as SFNSYEiGSv (SEQ ID NO:42), SFNSYEvGSi, (SEQ ID NO:43) SFNSYELGSv (SEQ ID NO:44), SFNSYELGSi (SEQ ID NO:45), SFNSYEiGSL (SEQ ID NO:46), SFNSYEvGSL (SEQ ID NO:47), aFNSYELGSL (SEQ ID NO:48), and any combination of the above-described modifications. Fragments and modification of fragments of δV1-1 are also contemplated, such as YELGSL (SEQ ID NO:49), YdLGSL (SEQ ID NO:50), ~~fDLGSL~~ fdLGSL (SEQ ID NO:51), ~~YDiGSL~~ YdiGSL ~~(SEQ I, iGSL~~ (SEQ ID ~~NO:59)D~~ NO:52), ~~YDvGSL~~ YdvGSL (SEQ ID NO:53), ~~YDLpsL~~ YdLpsL (SEQ ID NO:54), ~~YDLglL~~ YdLglL (SEQ ID NO:55), YdLGSi (SEQ ID NO:56), YdLGSv (SEQ ID NO:57), LGSL (SEQ ID NO:58), iGSL (SEQ ID NO:59), vGSL (SEQ ID NO:60), LpSL (SEQ ID NO:61), LGIL (SEQ ID NO:62), LGSi (SEQ ID NO:63), LGSv (SEQ ID NO:64).

Replace the paragraph on page 23, lines 11-12 with the following:

Accordingly, the term "a δPKC antagonist" as used herein intends a δPKC peptide, which refers to ~~any~~ a δV1-1 peptide, a δV1-2 peptide and a δV1-5 peptide.

Replace the paragraph on page 26, lines 11-19 with the following:

The plate is then incubated with a blocking solution (containing, for example bovine serum albumin) and then washed several times. A solution containing reporter-labelled (*e.g.*, radiolabelled ~~of~~ or fluorescently-tagged) peptide δV1-1 (SEQ ID NO: 4) and, in the test wells, as opposed to the control wells, a test compound is added. Different wells may contain different test compounds or different concentrations of the same test compound. Each test compound at each concentration is typically run in duplicate and each assay is typically run with negative (wells with no test compound) as well as positive (wells where the "test compound" is unlabeled peptide) controls. The free peptide is then washed out, and the degree of binding in the wells is assessed.

Replace the paragraph on page 26, lines 20-25 with the following:

A test compound is identified as active it if decreases the binding of the peptide, *i.e.*, if its effect on the ~~extend~~ extent of binding is above a threshold level. More specifically, if the decrease in binding is a several-fold different between the control and experimental samples, the compound would be considered as having binding activity. Typically, a 2-fold or 4-fold threshold difference in binding between the test and control samples is sought.